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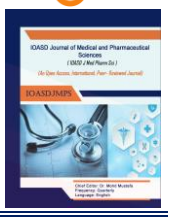

Hematologic Fallout: A Review of Anemia in Communities Exposed to Gas Flaring and Petrochemical Pollution

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ABSTRACT

Gas flaring and petrochemical pollution represent significant environmental hazards in oil-producing regions, particularly in low- and middle-income countries. These industrial activities release a wide array of toxic substances, including benzene, sulfur dioxide, and heavy metals, which contaminate air, soil, and water. Chronic exposure to these pollutants is increasingly associated with adverse hematologic effects, especially anemia. While anemia is often attributed to nutritional deficiencies and infectious diseases, growing evidence suggests that environmental exposures significantly contribute to its prevalence in vulnerable populations living near oil and gas facilities. This review examines the pathophysiological mechanisms through which gas flaring and petrochemical pollutants disrupt normal hematopoiesis. Key processes include bone marrow suppression by benzene metabolites, red blood cell destruction via oxidative stress, impaired iron metabolism due to inflammation, and reduced erythropoietin production from renal toxicity. Epidemiological studies from heavily industrialized regions such as the Niger Delta in Nigeria have reported disproportionately high rates of anemia among residents, particularly among children and pregnant women, further highlighting the intersection between environmental degradation and public health inequities.

Keywords: Gas flaring, Petrochemical pollution, Environmental anemia, Hematologic toxicity, Public health.

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ABBREVIATIONS

CO – Carbon monoxide
CO_2 – Carbon dioxide
EPO – Erythropoietin
Hb – Hemoglobin
HIF – Hypoxia-inducible factor
IL – Interleukin
IL-6 – Interleukin-6
LMICs – Low- and middle-income countries
NO_x – Nitrogen oxides
PAHs – Polycyclic aromatic hydrocarbons
PM – Particulate matter
$PM_{2.5}$ – Fine particulate matter with aerodynamic diameter ≤2.5 μm
ROS – Reactive oxygen species
SO_2 – Sulfur dioxide
WHO – World Health Organization

INTRODUCTION

Anemia remains a global public health issue, with an estimated 1.62 billion individuals affected worldwide. While commonly linked to iron deficiency, chronic disease, and parasitic infections, the role of environmental pollutants in the pathogenesis of anemia is increasingly gaining attention. In particular, communities residing near gas flaring sites and petrochemical industries face a growing burden of hematological disorders due to prolonged exposure to toxic emissions. Despite increasing global awareness, the full extent of the hematologic consequences of environmental pollution remains underexplored, especially in resource-limited regions where industrial activities intersect with socioeconomic vulnerability [1-2]. Gas flaring, the combustion of natural gas associated with oil extraction, is a routine practice in oil-producing regions such as the Niger Delta in Nigeria, parts of the Middle East, and Venezuela. This process releases a complex mixture of toxicants, including particulate

matter, volatile organic compounds (VOCs), heavy metals, and greenhouse gases. Similarly, petrochemical processing emits hazardous substances into the surrounding environment, contaminating the air, water, and soil. For residents living in close proximity to these operations, daily exposure to harmful pollutants is unavoidable and often unregulated [3-4].

Emerging evidence links these environmental exposures to a wide range of health complications, including respiratory diseases, cancers, reproductive dysfunction, and hematologic abnormalities. Anemia, defined by reduced hemoglobin levels or red blood cell mass, is among the most prevalent yet under-recognized outcomes. Studies have shown that toxins like benzene, lead, arsenic, and cadmium disrupt erythropoiesis through bone marrow suppression, hemolysis, and interference with iron metabolism. These pollutants not only affect red blood cell production but also accelerate their destruction, leading to a spectrum of anemia types—from microcytic and hypochromic to normocytic and hemolytic forms [5-6]. In gas-flaring zones, children and pregnant women appear to bear the heaviest burden of disease. Pediatric populations, whose hematopoietic systems are still developing, are particularly susceptible to the toxic effects of environmental contaminants. Prenatal exposure has been linked to low birth weight, prematurity, and neonatal anemia, with long-term developmental consequences. Women, especially during pregnancy, are at increased risk due to elevated iron demands and physiologic changes that heighten their vulnerability to environmental insults. Yet, health systems in these regions are often ill-equipped to monitor, diagnose, or manage environmentally induced hematologic conditions [7-8].

Compounding the health risks are socio-economic and infrastructural barriers. Communities near gas flaring sites often face limited access to clean water, nutritious food, and healthcare services—all of which further contribute to anemia prevalence. Moreover, a lack of environmental regulation and enforcement allows flaring and pollution to persist unchecked. The overlapping effects of environmental degradation, poverty, and weak health systems create a syndemic condition in which anemia thrives, disproportionately affecting marginalized populations [9].

AIM

This review aims to critically examine the impact of gas flaring and petrochemical pollution on anemia prevalence in exposed communities.

METHODS

This narrative review was conducted to synthesize current evidence on anemia in populations exposed to gas flaring and petrochemical pollution. A comprehensive literature search was performed across multiple databases, including PubMed, Scopus and Web of Science covering studies published up to 2025. Search terms combined key concepts such as *gas flaring*, *petrochemical pollution*, *anemia*, *hematotoxicity*, *oxidative stress*, and *heavy metals*. Eligible studies included cross-sectional, cohort, case-control, experimental, and review articles that examined hematologic outcomes in communities or populations with documented environmental exposure, with a focus on non-occupational cohorts to reflect population-level health effects. Articles not available in English or lacking primary data on anemia or hematologic parameters were excluded. Data extraction emphasized epidemiological findings, mechanistic pathways, clinical patterns, and environmental context. Relevant grey literature, governmental reports, and environmental assessments were also considered to contextualize exposure sources and community health impacts. Findings were synthesized narratively, integrating epidemiologic evidence with mechanistic insights to provide a comprehensive overview of the hematologic consequences of environmental pollution from gas flaring and petrochemical activities.

Sources and Composition of Pollution

The primary sources of environmental contamination in oil-producing regions are gas flaring and petrochemical processing. Gas flaring is the intentional burning of excess natural gas during oil extraction, often due to inadequate infrastructure for gas capture or commercial use. This practice is especially common in developing countries with weak regulatory enforcement, such as Nigeria, Angola, Iran, and parts of South America. Flaring typically occurs continuously near oil wells and produces both visible flames and invisible airborne pollutants that spread into surrounding communities [10]. In addition to gas flaring, petrochemical industries—which refine crude oil and process derivatives such as plastics, solvents, fertilizers, and synthetic rubbers—emit a wide array of hazardous pollutants. These operations often lack advanced emission control technologies and may discharge waste into nearby water bodies or unlined landfills. As a result, surrounding environments are contaminated via multiple pathways: airborne inhalation, ingestion of contaminated food and water, and dermal contact with polluted soil or surfaces [11].

The composition of pollutants released from gas flaring and petrochemical activities is diverse and highly toxic. Key pollutants include:

- **Volatile Organic Compounds (VOCs):** such as benzene, toluene, xylene, and ethylbenzene. Benzene, in particular, is a known hematotoxic and carcinogenic compound that impairs bone marrow function.
- **Polycyclic Aromatic Hydrocarbons (PAHs):** formed during incomplete combustion of hydrocarbons, PAHs are persistent environmental pollutants associated with DNA damage and oxidative stress.

- **Particulate Matter (PM2.5 and PM10):** tiny airborne particles that can enter the bloodstream through the respiratory tract, contributing to systemic inflammation and oxidative damage to red blood cells.
- **Heavy Metals:** including lead, arsenic, mercury, cadmium, and chromium. These metals accumulate in biological tissues and interfere with erythropoiesis, hemoglobin synthesis, and red blood cell survival.
- **Sulfur and Nitrogen Oxides (SO_2 and NO_x):** acidic gases that contribute to respiratory irritation and systemic inflammation, indirectly exacerbating anemia through chronic disease mechanisms.
- **Carbon Monoxide (CO):** a colorless, odorless gas that binds to hemoglobin more readily than oxygen, reducing oxygen transport capacity and potentially triggering hypoxic anemia [12].

These pollutants do not exist in isolation; they often co-exist in complex mixtures that have synergistic toxic effects, amplifying their impact on hematologic health. Moreover, prolonged low-dose exposure—common in residential areas near oil facilities—can be just as damaging as acute high-dose exposures, particularly in vulnerable groups such as children, the elderly, and individuals with pre-existing health conditions (Table 1) [13].

Table 1: Sources and Composition of Pollution from Gas Flaring and Petrochemical Activities

Source of Pollution	Primary Pollutants Released	Chemical Composition / Key Toxicants	Health-Relevant Characteristics
Gas Flaring	Combustion gases, particulate matter, volatile organic compounds (VOCs), polycyclic aromatic hydrocarbons (PAHs)	Carbon monoxide (CO), sulfur dioxide (SO_2), nitrogen oxides (NO_x), benzene, toluene, xylene, PAHs, particulate matter ($PM_{2.5}$, PM_{10})	Produces incomplete combustion products; rich in carcinogens and hematotoxins; contributes to air pollution and oxidative stress
Petrochemical Refinery Emissions	Fugitive VOC emissions, sulfur compounds, heavy metals, nitrogen oxides, particulate matter	Benzene, ethylbenzene, formaldehyde, hydrogen sulfide (H_2S), arsenic, lead, cadmium, nickel	VOCs and heavy metals have hematotoxic and genotoxic effects; contribute to anemia through bone marrow toxicity and oxidative damage
Oil Spills and Soil Contamination	Hydrocarbons, heavy metals, polyaromatic hydrocarbons (PAHs)	Crude oil components (alkanes, aromatics), lead, mercury, chromium, PAHs	Chronic soil contamination leads to bioaccumulation in food chains; ingestion and dermal exposure risk anemia via toxic effects
Wastewater Discharge	Hydrocarbon residues, heavy metals, chemical solvents	Benzene, toluene, xylene, lead, cadmium, mercury	Polluted water sources contribute to exposure through drinking and irrigation, impacting hematopoiesis and iron metabolism
Airborne Particulate Matter	Fine particles carrying adsorbed toxic chemicals	$PM_{2.5}$, PM_{10} loaded with heavy metals and organic toxins	Penetrates deep into lungs and systemic circulation; triggers inflammation and oxidative stress affecting red blood cells

Pathophysiological Mechanisms of Pollution-Induced Anemia

The development of anemia in individuals chronically exposed to gas flaring and petrochemical pollutants involves a multifaceted disruption of hematopoietic processes. These pollutants induce systemic toxicity through a combination of bone marrow suppression, oxidative stress, hemolysis, impaired iron metabolism, and inflammation, ultimately leading to insufficient red blood cell (RBC) production, increased destruction, or both [14]. One of the most well-documented mechanisms is bone marrow suppression, primarily associated with exposure to benzene, a volatile organic compound prevalent in both gas flaring and petrochemical emissions. Benzene and its metabolites, such as phenol and hydroquinone, interfere with hematopoietic stem cells in the bone marrow, leading to hypoplasia or aplasia of erythroid precursors. Chronic benzene exposure has been linked to a spectrum of hematologic abnormalities, including anemia, leukopenia, and thrombocytopenia, reflecting its broad marrow-toxic effects [15].

Oxidative stress is another major contributor to pollution-induced anemia. Pollutants such as polycyclic aromatic hydrocarbons (PAHs), particulate matter, and heavy metals (e.g., lead, arsenic, and cadmium) generate reactive oxygen species (ROS) that damage the membranes of erythrocytes, shorten their lifespan, and trigger premature hemolysis. This oxidative damage also impairs the function of enzymes like glucose-6-phosphate dehydrogenase (G6PD), rendering red cells

even more vulnerable to oxidative insults in genetically predisposed individuals [16]. Disruption of iron metabolism is another critical mechanism. Chronic exposure to inflammatory pollutants such as sulfur dioxide and nitrogen oxides can induce a state of low-grade systemic inflammation, which in turn elevates levels of hepcidin, a hepatic hormone that inhibits iron absorption and release from macrophages. This results in a form of anemia similar to anemia of chronic disease, where iron stores are adequate but functionally inaccessible for erythropoiesis. Concurrent nutritional deficiencies, common in impoverished communities near flare sites, further compound this imbalance [17-18].

Furthermore, nephrotoxicity caused by pollutants such as mercury and cadmium can impair renal erythropoietin production, which is essential for stimulating red blood cell production in the bone marrow. Reduced erythropoietin levels lead to normocytic normochromic anemia, especially in individuals with pre-existing renal vulnerability or comorbidities like hypertension and diabetes [19]. In pregnant women, pollutant exposure can interfere with placental oxygen transport and iron transfer, increasing the risk of fetal anemia, low birth weight, and developmental delays. Children exposed in utero or during early life may experience long-lasting hematologic effects due to both direct toxic insult and epigenetic changes affecting gene expression in erythropoiesis-related pathways (Table 2) [20].

Table 2: Pathophysiological Mechanisms of Pollution-Induced Anemia

Mechanism	Description	Key Pollutants Involved	Hematologic Impact
Oxidative Stress and Inflammation	Pollutants generate reactive oxygen species (ROS), triggering chronic inflammation and oxidative damage to erythrocytes and bone marrow cells.	Benzene, PAHs, heavy metals (lead, cadmium)	Increased red blood cell (RBC) destruction; impaired erythropoiesis
Bone Marrow Suppression	Toxic substances inhibit proliferation and differentiation of hematopoietic stem cells, reducing RBC production.	Benzene, heavy metals, hydrocarbons	Reduced red blood cell count; aplastic anemia in severe cases
Disrupted Iron Metabolism	Pollutants interfere with iron absorption, transport, and storage, leading to functional iron deficiency.	Lead, cadmium, arsenic	Impaired hemoglobin synthesis and ineffective erythropoiesis
Hemolysis	Direct oxidative damage or immune-mediated destruction of erythrocytes caused by exposure to toxicants.	Sulfur dioxide, benzene derivatives	Shortened RBC lifespan, increased anemia severity
Nutritional Deficiencies	Pollution-related gastrointestinal disturbances and food contamination result in poor nutritional status, especially iron and vitamin deficiencies.	Various petrochemical contaminants	Exacerbates anemia through inadequate substrate for RBC production
Genotoxicity and DNA Damage	Pollutants cause DNA strand breaks and mutations in hematopoietic cells, affecting cell cycle and survival.	Benzene, PAHs	Bone marrow dysfunction, risk of hematologic malignancies

Epidemiological Evidence from Affected Communities

A growing body of epidemiological research highlights a concerning correlation between environmental exposure to gas flaring and petrochemical pollution and the prevalence of anemia in surrounding populations. However, much of this evidence remains region-specific, fragmented, and often underreported due to limited surveillance infrastructure and inadequate environmental health monitoring in low- and middle-income countries [21]. In the Niger Delta region of Nigeria, one of the most studied gas flaring hotspots, several cross-sectional and observational studies have documented elevated rates of anemia among residents, particularly children and women of reproductive age. For instance, a community-based study conducted in Bayelsa State found that over 65% of children under five years living within five kilometers of active gas flaring sites were anemic, with significantly lower hemoglobin levels compared to children in non-flaring areas. Similarly, adult residents in flaring zones reported higher incidences of fatigue, pallor, and dizziness—clinical signs often consistent with moderate to severe anemia. Biomonitoring revealed elevated blood lead and benzene metabolite levels in exposed individuals, correlating with reduced hematocrit and RBC counts [22-23].

Epidemiologic investigations in other oil-producing nations such as Iran, Venezuela, and parts of the Arabian Gulf echo similar patterns. In Mahshahr, Iran—home to one of the country's largest petrochemical complexes—a comparative study found significantly higher rates of anemia and leukopenia among refinery workers and nearby residents than in populations living farther away. The findings were attributed to chronic exposure to benzene and sulfur dioxide, both of which have documented hematotoxic effects. Likewise, studies from industrial towns in eastern Venezuela showed a

positive association between proximity to petrochemical plants and abnormal complete blood count (CBC) parameters, including reduced hemoglobin concentration and elevated red cell distribution width (RDW), a marker of anisocytosis commonly observed in toxic or inflammatory anemias [24-25]. Children are disproportionately affected across various contexts. A study in Ecuador's Amazon region, where flaring and crude oil contamination are rampant, found that school-aged children exposed to petrochemical by-products had significantly lower hemoglobin levels and higher urinary concentrations of PAH metabolites. Moreover, evidence suggests that even prenatal exposure can alter fetal hematologic profiles; maternal residence near flare sites has been linked to low cord blood hemoglobin and iron indices, raising concerns about long-term developmental and cognitive impacts [26-27].

Vulnerable Populations and Socioeconomic Implications

The hematologic consequences of environmental pollution are not uniformly distributed across populations. Instead, they disproportionately affect vulnerable groups—notably children, pregnant women, the elderly, and economically marginalized communities—who often live closest to gas flaring sites and petrochemical installations due to systemic inequalities and lack of political agency. These populations are doubly burdened: first by heightened biological susceptibility to anemia, and second by limited access to healthcare and social support systems needed for diagnosis and treatment [28-29]. Children are particularly sensitive to the hematotoxic effects of pollutants due to their rapidly developing systems and higher relative intake of air, food, and water. Exposure to lead, benzene, and particulate matter has been associated with reduced hemoglobin synthesis, impaired cognitive development, stunted growth, and increased school absenteeism. The consequences of chronic anemia in childhood are far-reaching, contributing to intergenerational cycles of poverty due to poor educational performance and diminished future economic productivity [30].

Pregnant women in polluted environments face heightened risks of iron deficiency, gestational anemia, and adverse birth outcomes. Hematologic insufficiency during pregnancy is associated with an increased likelihood of preterm birth, low birth weight, and perinatal mortality. Additionally, polluted air and contaminated water supplies compromise maternal nutrition and immune function, further exacerbating anemia severity. These health threats are magnified in settings where antenatal care is inadequate or inaccessible, making early detection and intervention difficult [31]. Beyond biological vulnerability, socioeconomic status plays a critical role in mediating both exposure and outcome. Many pollution-exposed communities reside in resource-poor rural or peri-urban zones, where healthcare infrastructure is weak, environmental regulation is minimal, and economic dependency on oil and petrochemical industries discourages local activism. These populations often lack formal employment, access to clean water, and educational resources that could empower them to demand safer living conditions or seek timely medical care [32].

The economic implications of widespread, pollution-induced anemia are profound. Reduced labor capacity due to fatigue and illness affects productivity in agriculture, small-scale trading, and informal work sectors, which dominate the livelihoods of many in oil-rich regions. Additionally, the direct and indirect costs of managing anemia—ranging from diagnostic testing and iron supplementation to hospitalization for severe complications—place a significant financial burden on already impoverished households. In areas without universal health coverage, out-of-pocket expenditures can push families deeper into poverty [33]. At a societal level, the cumulative impact of untreated anemia contributes to lower community resilience, reduced educational attainment, weakened workforce potential, and strained public health systems. Over time, this erodes human capital development and perpetuates inequities, particularly in regions already marginalized by national policy and corporate exploitation [34-35].

Current Gaps in Policy and Intervention

Despite mounting evidence linking gas flaring and petrochemical pollution to adverse hematologic outcomes, policy frameworks and intervention strategies remain inadequate, fragmented, and poorly enforced, particularly in low- and middle-income countries. These gaps stem from a combination of regulatory inertia, lack of political will, insufficient environmental health data, and competing economic interests, especially in oil-dependent economies [36]. One of the most significant policy gaps lies in the weak enforcement of environmental protection laws. In many oil-producing nations, regulations governing gas flaring, emission thresholds, and waste disposal are either outdated or circumvented through legal loopholes and poor monitoring. Even where legislation exists—such as Nigeria's Gas Flaring (Prohibition and Punishment) Act—the absence of stringent implementation mechanisms and accountability has allowed polluting activities to persist unabated, often in close proximity to residential areas. This failure to regulate emissions perpetuates chronic exposure, with no recourse for affected communities [37].

Furthermore, environmental health surveillance systems are grossly underdeveloped. Many regions affected by petrochemical activities lack the infrastructure to monitor air, water, and soil quality in real time, let alone assess individual exposure levels. Without reliable data, it becomes nearly impossible to establish causal relationships between pollution and anemia or to prioritize communities for intervention. The

absence of longitudinal epidemiological studies further weakens the evidence base needed to inform policy, leaving public health authorities ill-equipped to respond [38]. On the healthcare front, screening and treatment programs for anemia rarely account for environmental etiologies. Anemia management strategies in affected regions often focus narrowly on nutritional supplementation or parasitic infections, overlooking the potential role of chronic toxic exposures. This biomedical tunnel vision results in repeated treatment failures and chronic underdiagnosis of environmentally induced anemia. Health workers are seldom trained to recognize the signs of toxic hematopathy, and public awareness campaigns addressing the health risks of gas flaring are minimal to nonexistent [39].

Another critical gap is the lack of intersectoral coordination. Environmental health issues often fall into the grey area between ministries of health, environment, energy, and labor—resulting in fragmented responsibilities and diluted accountability. This bureaucratic fragmentation impedes the development of comprehensive action plans that address both the health and environmental dimensions of petrochemical pollution. The absence of community participation in policy design and enforcement also contributes to low trust in governmental interventions and limited grassroots advocacy [40]. Economic and political interests further complicate reform efforts. In countries where oil and petrochemical industries are major contributors to GDP, government-corporate entanglements frequently stifle regulatory action. Polluting companies often enjoy tax incentives, political protection, or operate under joint ventures with national governments, creating conflicts of interest that undermine environmental justice [41]. There is a marked lack of international attention and support. Global health initiatives and donor programs have traditionally prioritized infectious diseases, maternal health, and malnutrition in low-resource settings, leaving environmental hematology an overlooked domain. There is an urgent need for global frameworks, funding mechanisms, and partnerships to support national efforts in combating pollution-induced hematologic disorders.

Recommendations

Addressing the multifaceted challenge of anemia in communities exposed to gas flaring and petrochemical pollution demands a multisectoral, evidence-based, and community-centered approach. The following recommendations aim to bridge existing gaps in policy, research, and public health interventions to mitigate hematologic fallout and improve population health outcomes.

1. **Strengthen Environmental Regulations and Enforcement:**

Governments must update and rigorously enforce environmental laws to limit gas flaring and toxic emissions from petrochemical operations. This includes establishing stringent emission standards, regular environmental audits, and imposing substantial penalties for violations. Transparent monitoring systems using modern technology such as remote sensing and real-time air quality measurement should be institutionalized to ensure compliance and empower communities with actionable data.

2. **Expand Environmental Health Surveillance and Research:**

Investment in comprehensive environmental health surveillance is crucial to capture accurate exposure data and hematologic outcomes over time. Longitudinal cohort studies, biomonitoring, and integrated health-environment databases will provide the robust evidence needed to inform targeted interventions and refine risk assessments. Collaboration between academia, government agencies, and international organizations can facilitate resource sharing and capacity building.

3. **Integrate Environmental Factors into Anemia Screening and Management:**

Healthcare systems in affected regions should incorporate environmental exposure histories into routine anemia screening protocols. Training healthcare providers to recognize pollution-related hematologic disorders will improve diagnosis and guide appropriate management, including chelation therapy where relevant. Public health programs should address both nutritional deficiencies and toxic exposures to ensure comprehensive care.

4. **Empower Vulnerable Communities through Education and Participation:**

Community education initiatives must raise awareness about the health risks of gas flaring and petrochemical pollution and promote protective behaviors. Inclusive policy development should engage community representatives, local leaders, and civil society to foster trust, enhance advocacy, and ensure interventions are culturally appropriate and equitable. Empowering communities strengthens accountability mechanisms and supports environmental justice.

5. **Foster Intersectoral Collaboration and Policy Integration:**

A coordinated approach involving ministries of health, environment, energy, labor, and finance is essential to harmonize regulations and streamline response efforts. Establishing multi-agency task forces and cross-sectoral frameworks can facilitate integrated planning, resource allocation, and monitoring. International cooperation and partnerships should be leveraged to align national policies with global environmental and health agendas.

6. **Promote Economic Diversification and Sustainable Development:**

Reducing dependence on oil and petrochemical industries through economic diversification can alleviate

political and financial barriers to environmental reform. Investments in renewable energy, agriculture, and small enterprises may improve community resilience, reduce pollution sources, and support sustainable livelihoods, thereby indirectly improving hematologic health.

7. Advocate for Global Recognition and Funding:

Environmental hematology should gain greater visibility in global health priorities. International donors, development agencies, and research funders need to allocate resources specifically for addressing pollution-induced anemia and related disorders. Global frameworks should facilitate knowledge exchange, technical assistance, and financial support to countries grappling with the dual burden of environmental degradation and poor health.

CONCLUSION

Anemia in communities exposed to gas flaring and petrochemical pollution represents a significant but often overlooked public health crisis. The confluence of toxic environmental exposures, socioeconomic vulnerabilities, and inadequate healthcare responses exacerbates the burden of hematologic disorders, particularly among children, pregnant women, and marginalized populations. Epidemiological evidence consistently points to a strong association between chronic exposure to petrochemical pollutants and the development of anemia through complex pathophysiological mechanisms, including oxidative stress, bone marrow suppression, and disrupted iron metabolism. Current policies and interventions fall short in addressing this challenge, hindered by weak regulatory enforcement, insufficient environmental and health surveillance, fragmented institutional coordination, and political-economic conflicts of interest. Moreover, the lack of integration of environmental factors into anemia management and the minimal involvement of affected communities further undermine effective mitigation efforts. These gaps contribute to ongoing health inequities and perpetuate the cycle of poverty and illness in affected regions.

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